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Comparison of Mitral Inflow and Superior Vena Cava Doppler Velocities in Chronic Obstructive Pulmonary Disease and Constrictive Pericarditis

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Objective. **This study was conducted to determine whether Doppler recording of superior vena cava flow velocities can differentiate chronic obstructive pulmonary disease from constrictive pericarditis in patients with a respiratory variation of ≥25% in mitral inflow E velocity.**

Background. **Although respiratory variation (≥25%) in mitral E velocity is the main diagnostic criterion for constrictive pericarditis by Doppler echocardiography, it can also be present in chronic obstructive pulmonary disease. Because the respiratory variation is due to increased change in intrathoracic pressure with respiration in chronic obstructive pulmonary disease, and to dissociation of intrathoracic-intracardiac pressure changes in constriction, it was hypothesized that the Doppler flow velocity pattern in the superior vena cava (affected by intrathoracic pressure) would be different in these two conditions.**

Methods. **Pulsed-wave Doppler recording of mitral and superior vena cava flow velocities in 20 patients with chronic obstructive pulmonary disease who had ≥25% respiratory variation in mitral E-wave velocity were compared with those of 20 patients who had surgically proved constrictive pericarditis.**

Results. **Constrictive pericarditis and chronic obstructive pulmonary disease had similar respiratory variation in mitral E velocity (41% versus 46%). In the latter, the E/A ratio was lower (inspiration, 0.8 ± 0.3 versus 1.5 ± 0.7 [$p < 0.0001$]; expiration, 1.0 ± 0.3 vs. 1.9 ± 0.7 [$p < 0.0001$]) and deceleration time longer (inspiration, 198 ± 53 ms versus 137 ± 32 ms; expiration, 225 ± 43 ms vs. 161 ± 33 ms [$p < 0.0001$]). Inspiratory superior vena cava systolic forward flow velocity was significantly higher in chronic obstructive pulmonary disease (72.9 ± 22.6 cm/s versus 36.2 ± 9.3 cm/s, $p < 0.0001$), while expiratory systolic forward flow velocity was similar. Hence, there was a significantly greater respiratory variation in superior vena cava systolic forward flow velocity in chronic obstructive pulmonary disease without an overlap with constrictive pericarditis (39.5 ± 18.8 cm/s vs. 4.2 ± 3.4 cm/s, $p < 0.0001$).**

Conclusions. **Despite a similar respiratory variation in mitral E wave velocities, mitral inflow variables in chronic obstructive pulmonary disease are less restrictive compared with those in constrictive pericarditis. More importantly, patients with chronic obstructive pulmonary disease show a marked increase in inspiratory superior vena cava systolic forward flow velocity, which is not seen in patients with constrictive pericarditis.**

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Respiratory variation of ≥25% in Doppler mitral E wave velocity is a well-recognized diagnostic feature of constrictive pericarditis that is useful in distinguishing pericardial constriction from restrictive cardiomyopathy (1,2). However, this respiratory variation is also found in other diseases, especially chronic obstructive pulmonary disease. Because patients with constrictive pericarditis and chronic obstructive pulmonary disease can have similar clinical presentations, for example, dyspnea and right-sided heart failure, a reliable way to differentiate between these two conditions would be helpful. The respiratory variation of mitral E velocity in chronic obstructive pulmonary disease is related to exaggerated swings in intrathoracic pressure with respiration (3), whereas in constrictive pericarditis it is believed to be related to the thickened pericardium, which prevents full transmission of intrathoracic pressure changes to the cardiac chambers (1). Because flow velocities in the superior vena cava are affected by the right atrial pressure changes with respiration (4), patients with chronic obstructive pulmonary disease should have a marked inspiratory increase in superior vena cava forward Doppler velocity that would not be present in patients with constrictive pericarditis. To test this hypothesis, we designed a study to assess the value of superior vena cava Doppler in differentiating chronic obstructive pulmonary disease from constrictive pericarditis when the respiratory variation in mitral E velocity was ≥25%.

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Abbreviations and Acronyms

FEV = forced expiratory volume in 1 s
FVC = forced vital capacity

Methods

Patients. Twenty patients (11 males and 9 females) with chronic obstructive pulmonary disease who were in sinus rhythm and had ≥25% respiratory variation in mitral early diastolic filling velocity (E wave) were studied. The diagnosis of chronic obstructive pulmonary disease was based on the clinical history and pulmonary function test (standard spirometry) results, that is, forced expiratory volume in 1 s (FEV_1) <70% of predicted value or the ratio of FEV_1 to forced vital capacity (FVC) <0.70, or both. The Doppler echocardiographic measurements of mitral and superior vena cava flow velocities were compared with those of 20 patients (19 males and 1 female) who subsequently had surgically confirmed constrictive pericarditis. The patients with constrictive pericarditis were selected from the data base on the basis of being in sinus rhythm, having respiratory changes in mitral E velocity of ≥25%, and having superior vena cava Doppler recording for the analysis. Two patients in the constrictive pericarditis group had concomitant chronic obstructive pulmonary disease.

Echocardiography. All examinations were performed with a commercially available cardiac ultrasonographic instrument, using a 2.5- or 3.5-MHz transducer. Pulsed-wave Doppler echocardiography was performed with simultaneous respiratory recording with a nasal respirometer. Mitral flow velocities were recorded from the apical window, with a 1- to 3-mm sample volume placed between the tips of the mitral leaflets during diastole. Superior vena cava flow velocities were measured from the right supraclavicular fossa or suprasternal notch. The sample volume size was 1 to 3 mm and the mean depth of the sample volume was 5.5 ± 0.3 cm. The echocardiogram and Doppler velocities were recorded on videotape at recording speeds of 25, 50 and 100 mm/s.

Analysis of Doppler flow velocities. All the Doppler measurements were performed manually on the still frame of the videotape by using a built-in calculation package. Analysis of all Doppler velocities was performed on the first cardiac cycle after the onset of inspiration and expiration, and the values were averaged from three respiratory cycles. From the mitral Doppler tracing, the following variables were measured: the peak velocity of early diastolic filling (E wave) and late filling with atrial contraction (A wave), the E/A ratio and the deceleration time of the E wave. From the superior vena cava Doppler study, peak velocities and flow velocity integrals of systolic forward, diastolic forward, end-systolic reversal and atrial reversal waves were measured.

Statistical analysis. The effects of patient group (i.e., chronic obstructive pulmonary disease vs. constrictive pericarditis) and respiratory phase on the echocardiographic parameters were investigated within the general linear mixed model framework. In these analyses, the fixed portion of the model consisted of the main effects for patient group and respiratory phase as well as the interaction between these two main effects. The interaction effect was included to test whether the changes in echocardiographic parameter between inspiration and expiration phases were consistent between the chronic obstructive pulmonary disease and constrictive pericarditis patient groups. To account for the possible correlation in inspiration and expiration measurements, a random effect for subject was incorporated into the model. Comparisons between patient groups during inspiration or expiration were investigated within the above general linear mixed model. All continuous variables are expressed as mean ± 1 SD. The percentage difference between the inspiratory and expiratory mitral Doppler measurements was calculated as a percent increase relative to inspiration.

Results

Clinical characteristics. The mean age of the patients with chronic obstructive pulmonary disease (63 ± 10 years) and constrictive pericarditis (58 ± 12 years) was similar ($p = 0.29$). The mean heart rate was 75 ± 8 beats/min for those with chronic obstructive pulmonary disease and 77 ± 10 beats/min for those with constrictive pericarditis ($p = 0.25$); the mean respiratory rate was 20 ± 4/min and 19 ± 4/min ($p = 0.93$), respectively; and mean blood pressure was 101 ± 13 mm Hg and 89 ± 12 mm Hg, respectively ($p = 0.0015$).

The causes of chronic obstructive pulmonary disease were chronic cigarette smoking in 18 patients, $alpha_1$-antitrypsin deficiency in 1 and occupational lung disease in one. Three of the patients with a history of cigarette smoking had concomitant sleep apnea and one had previous pneumonectomy because of pulmonary tuberculosis. Pulmonary function tests in the group with chronic obstructive pulmonary disease showed a mean FEV_1 of 29% ± 16% (range, 14% to 53%) of the predicted value and an FEV_1/FVC of 0.41 ± 0.18 (range, 0.19 to 0.69). Six patients with chronic obstructive pulmonary disease underwent computed tomography of the chest, and no pericardial thickening or calcification was noted.

The causes of constrictive pericarditis were idiopathic in nine patients, possible viral pericarditis in five, previous cardiac surgery in four, posttraumatic hemopericardium (after pacemaker implantation) in one and rheumatoid arthritis in one.

Mitral doppler measurements. Table 1 compares the mitral Doppler measurements for the chronic obstructive pulmonary disease and constrictive pericarditis patient groups. There were no significant interactive effects of patient group and respiratory phase for E, A and deceleration time, indicating that respiratory changes in these parameters were consistent between the patient groups ($p = 0.91, 0.29, 0.82$, respectively) (Fig. 1); percent E velocity change was 46% and 41%, A velocity change was 9% and 7% and deceleration time change was 15% and 19% for the chronic obstructive pulmonary disease and constrictive pericarditis groups, respectively. There was no significant difference between patient groups in regard

Table 1. Comparison of Mitral Doppler Measurements in Chronic Obstructive Pulmonary Disease and Constrictive Pericarditis

Variable	Chronic Obstructive Pulmonary Disease	Constrictive Pericarditis	p
Inspiration			
E velocity (cm/s)	52.3 ± 15.8*	64.9 ± 25.5*	0.1013
A velocity (cm/s)	71.2 ± 16.6†	47.3 ± 21.3	0.0005
E/A ratio	0.8 ± 0.3*	1.5 ± 0.6*	< 0.0001
Deceleration time (ms)	201 ± 52*	137 ± 32*	< 0.0001
Expiration			
E velocity (cm/s)	75.2 ± 22.5*	88.2 ± 29.2*	0.0928
A velocity (cm/s)	76.7 ± 17.5†	50.4 ± 22.8	0.0001
E/A ratio	1.0 ± 0.3*	1.9 ± 0.7*	< 0.0001
Deceleration time (ms)	226 ± 43*	161 ± 33*	< 0.0001

Values are mean ± SD. *$p < 0.0001$ inspiration vs. expiration. †$p < 0.01$ inspiration vs. expiration.

to E velocity ($p = 0.09$). However, there were significant differences between patient groups in regard to A velocity ($p = 0.0002$) and deceleration time ($p = 0.0001$). The mitral E deceleration time was shorter in the constrictive pericarditis group (inspiration 137 ± 32 ms vs. 198 ± 53 ms, $p < 0.0001$; expiration 161 ± 33 ms vs. 225 ± 43 ms, $p < 0.0001$).

Superior vena cava Doppler measurements. The velocities and flow velocity intervals of the two groups of patients are shown in Tables 2 and 3. There were significant interactive effects of patient group and respiratory phase for systolic forward flow ($p = 0.0001$) and diastolic forward flow ($p = 0.0001$) velocities. The inspiratory systolic forward flow velocity was higher in the group with chronic obstructive pulmonary disease (72.9 ± 22.6 cm/s vs. 36.2 ± 9.3 cm/s), whereas the inspiratory diastolic forward flow velocity was similar (Fig. 2). The expiratory diastolic forward flow velocity was significantly lower in the chronic obstructive pulmonary disease group (15.2 ± 12.8 cm/s vs. 32.5 ± 14.0 cm/s, $p < 0.0018$). The absolute change in systolic and diastolic forward flow velocities between inspiration and expiration was significantly higher in the group with chronic obstructive pulmonary disease (inspiratory-expiratory systolic forward flow velocity, 39.5 ± 18.8 cm/s vs. 4.2 ± 3.4 cm/s [$p < 0.0001$]; inspiratory-expiratory diastolic forward flow velocity, 30.3 ± 20.1 vs. 7.5 ± 8.6 [$p < 0.0001$]) (Fig. 3). Although the difference of inspiratory-expiratory diastolic forward flow velocity was highly significant, there was a considerable overlap between the two groups. In contrast, the inspiratory-expiratory systolic forward flow velocity showed a minimum overlap between the two groups.

Figure 1. Mitral inflow Doppler from patients with chronic obstructive pulmonary disease (COPD) **(top)** or constrictive pericarditis **(bottom)** showing respiratory variation in mitral E velocity **(arrows).** ins, inspiration; exp, expiration.

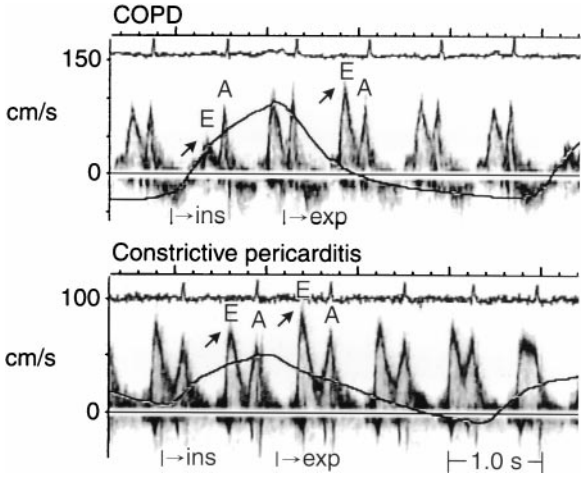

Table 2. Comparison of Superior Vena Cava Peak Forward and Reverse Flow Velocity in Chronic Obstructive Pulmonary Disease and Constrictive Pericarditis

Peak Velocity	Chronic Obstructive Pulmonary Disease	Constrictive Pericarditis	p
Inspiration (cm/s)			
Systolic forward flow	72.9 ± 22.6	36.2 ± 9.3	< 0.0001
Diastolic forward flow	45.5 ± 20.3	40.0 ± 17.4	0.2952
End-systolic reversal	2.5 ± 6.2	7.3 ± 8.6	0.0700
Atrial reversal	17.9 ± 7.1	12.4 ± 7.8	0.0190
Expiration (cm/s)			
Systolic forward flow	33.5 ± 12.8	32.1 ± 8.0	0.7597
Diastolic forward flow	15.2 ± 12.7	32.5 ± 14.0	< 0.0018
End-systolic reversal	7.7 ± 9.2	8.3 ± 8.3	0.8318
Atrial reversal	16.6 ± 7.2	12.5 ± 5.9	0.0730
Inspiration-expiration (cm/s)			
Systolic forward flow	39.4 ± 18.8	4.1 ± 3.5	< 0.0001
Diastolic forward flow	30.3 ± 20.0	7.5 ± 8.6	< 0.0001
End-systolic reversal	−5.2 ± 9.4	−1.0 ± 6.1	0.0977
Atrial reversal	1.23 ± 10.3	−0.1 ± 3.93	0.5867

Values are mean ± SD.

Table 3. Comparison of Superior Vena Cava Forward and Reverse Flow Velocity Integrals in Chronic Obstructive Pulmonary Disease and Constrictive Pericarditis

Flow Velocity Integrals	Chronic Obstructive Pulmonary Disease	Constrictive Pericarditis	p
Inspiration (cm)			
Systolic forward flow	20.2 ± 7.7	6.2 ± 2.3	< 0.0001
Diastolic forward flow	7.6 ± 3.8	6.2 ± 2.5	0.1700
End-systolic reversal	0.2 ± 0.6	0.7 ± 0.9	0.0955
Atrial reversal	1.9 ± 1.2	1.2 ± 0.9	0.0247
Expiration (cm)			
Systolic forward flow	8.2 ± 4.0	5.1 ± 1.9	0.0373
Diastolic forward flow	3.0 ± 3.4	4.9 ± 2.3	0.0595
End-systolic reversal	0.8 ± 1.0	0.7 ± 0.9	0.8193
Atrial reversal	1.5 ± 0.7	1.3 ± 0.8	0.5587
Inspiration-expiration (cm)			
Systolic forward flow	12.0 ± 7.1	1.2 ± 1.2	< 0.0001
Diastolic forward flow	4.5 ± 6.0	1.3 ± 1.3	0.0228
End-systolic reversal	−0.5 ± 1.0	0 ± 0.5	0.0402
Atrial reversal	0.5 ± 1.6	0 ± 0.5	0.1706

Values are mean ± SD.

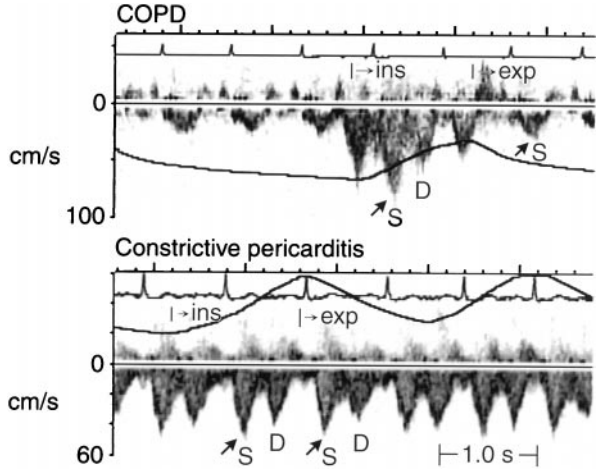

Figure 2. Superior vena cava Doppler from a patient with constrictive pericarditis shows little respiratory changes in systolic forward flow velocity **(bottom)** from inspiration to expiration **(arrows)**, in contrast to marked phasic inspiratory augmentation of forward flow velocity in chronic obstructive pulmonary disease (COPD) **(top)**. S, systolic forward flow; D, diastolic forward flow; ins, inspiration; exp, expiration.

Discussion

Hemodynamics and Doppler echocardiographic features of constrictive pericarditis and chronic obstructive pulmonary disease. The echocardiographic diagnosis of constrictive pericarditis has been enhanced by incorporating the characteristic Doppler feature of respiratory variation of mitral inflow E velocity, as described by Hatle et al. (1). However, other conditions can cause a similar respiratory variation in the Doppler mitral E wave velocity, including cardiac tamponade (5), acute right ventricular dilatation due to right ventricular infarction or pulmonary embolism and chronic obstructive pulmonary disease (1). Most of these conditions can be distinguished by clinical and morphological echocardiographic features (i.e., presence of a pericardial effusion or markedly dilated right ventricle). However, it often is difficult to distinguish chronic obstructive pulmonary disease from constriction when they have similar clinical presentations. Hoit et al. (3) reported increased respiratory Doppler variation in mitral and tricuspid flow in chronic lung disease, and emphasized the importance of identifying this condition as a cause of such respiratory change in Doppler flow velocities to avoid unnecessary investigation and procedures. Oh et al. (2) reported the diagnostic role of Doppler echocardiography in constrictive pericarditis. In that study of 23 patients with preoperative Doppler echocardiographic findings of constrictive pericarditis, 22 patients had constrictive pericarditis confirmed intraoperatively. One patient with normal pericardium at surgery had severe chronic obstructive pulmonary disease that might have been the cause of the false-positive Doppler diagnosis of constriction.

The mechanism for respiratory variation in Doppler mitral E wave velocity in these two groups of patients is different. Under normal circumstances, the inspiratory fall in intrathoracic pressure is accompanied by a decrease in intrapericardial and intracardiac pressures of similar magnitude. However, in constrictive pericarditis, there is a dissociation of intrapleural and intracardiac pressures thought to be due to a thickened and sometimes calcified pericardium, which prevents full transmission of intrathoracic pressure changes with respiration to the cardiac chambers (1). As a result, the pressure gradient, hence, flow velocity, from the pulmonary veins to the left atrium and to the left ventricle is decreased with inspiration.

Airway obstruction with increased respiratory effort can exaggerate the decrease in systemic blood pressure and result in pulsus paradoxus during inspiration (6). It is postulated that the exaggerated swings in intrathoracic pressures with respiration are the cause of such findings. Blaustein et al. (7) demonstrated that during induced bronchospasm or increased resistance to breathing, intrapleural pressure becomes more negative during inspiration and less negative or even positive during expiration. The more negative inspiratory intrapleural pressure results in two consequences. First, increased venous return to the right cardiac chambers shifts the interventricular septum to the left and decreases left ventricular end-diastolic

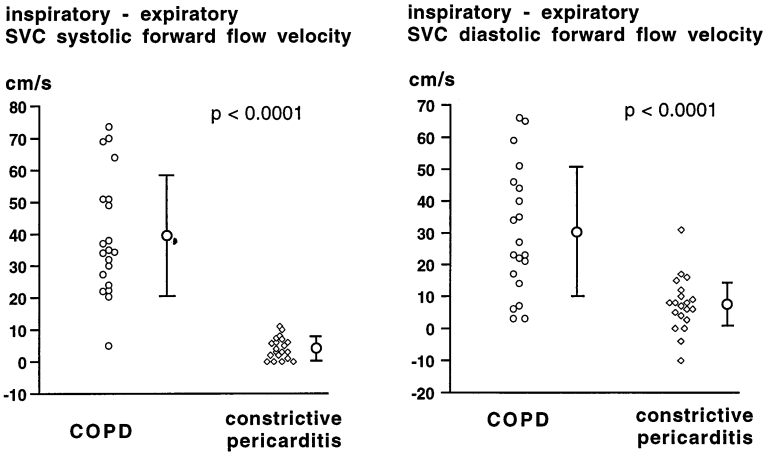

Figure 3. Difference between inspiratory and expiratory systolic forward flow velocity **(left)** and diastolic forward flow velocity **(right)** in chronic obstructive pulmonary disease (COPD) and constrictive pericarditis patients. SVC, superior vena cava.

volume and compliance (8). Similar changes together with respiratory mitral and tricuspid Doppler E velocity variation have been observed in patients with sleep apnea during the episodes of sleep-associated airway obstruction (9). Second, the negative intrapleural pressure also increases left ventricular afterload by increasing aortic transmural pressure (10). This may also result in respiratory changes in left ventricular output and pressure.

The superior vena cava is an intrathoracic structure, and its flow velocities have been documented to correlate with right atrial pressure (11). Hence, Doppler echocardiographic recording from the superior vena cava usually shows an increase in the forward flow velocity during inspiration (12). Because restriction to cardiac filling and dissociation of intrapleural and intracardiac pressure causes right atrial pressure to remain increased and constant throughout the respiratory cycle in constrictive pericarditis (13), the respiratory change in superior vena cava forward flow velocity is minimal (14,15). However, in patients with chronic obstructive pulmonary disease, increased swings in intrapleural pressure cause the right atrial pressure to decrease more than usual during inspiration, which results in augmentation of superior vena cava forward flow velocities toward the right atrium. As demonstrated by Izumi et al. (16), patients with pulmonary disease showed significantly more negative pleural pressure during inspiration in comparison with healthy subjects, and the velocity of systolic and diastolic forward flow during inspiration in patients with pulmonary disease was significantly higher than that of normal subjects. In the present study, all 20 patients with constrictive pericarditis had minimal respiratory variation in the superior vena cava systolic and diastolic forward flow velocities. In comparison, 19 of the 20 patients (95%) with chronic obstructive pulmonary disease had respiratory variation in systolic forward flow velocity of ≥20 cm/s, or greater than 35% increase in the velocity with inspiration. The patient whose systolic forward velocity had a respiratory variation <20 cm/s (5 cm/s) had paradoxical diaphragmatic motion due to very severe lung hyperinflation. In a study of patients with severe pulmonary emphysema, the correlation between transdiaphragmatic pressure (abdominal pressure minus pleural pressure) and respiratory variation of the superior vena cava Doppler systolic forward flow velocity was high ($r = 0.88$, $p = 0.0002$) (17). When the severity of chronic obstructive pulmonary disease, especially pulmonary emphysema, is very far advanced and diaphragmatic dysfunction develops, the respiratory variation in superior vena cava systolic forward flow velocity will become less than expected.

Conclusions

In patients with chronic obstructive pulmonary disease, respiratory variation of the mitral E velocity may be similar in magnitude to that of patients with constrictive pericarditis. Although patients with constrictive pericarditis are more likely to exhibit restrictive mitral inflow characteristics (E/A >1.5 and deceleration time <160 ms) than patients with chronic obstructive pulmonary disease, there is sufficient overlap that these variables cannot be used alone in individual cases. In contrast, the superior vena cava Doppler flow velocity pattern is distinctly different between chronic obstructive pulmonary disease and constrictive pericarditis, with constrictive pericarditis patients showing minimal augmentation of superior vena cava inspiratory flow, and those with chronic obstructive pulmonary disease showing exaggerated increases in inspiratory flow. This variable, especially respiratory variation of systolic forward flow velocity, shows minimal overlap between the two diseases, with the rare exception of patients with such hyperinflated lungs that there is paradoxical diaphragmatic motion. Therefore, recording of superior vena cava Doppler flow velocities should be an essential part of a complete Doppler echocardiographic investigation of constrictive pericarditis. The finding of significant inspiratory accentuation of superior vena cava forward flow velocities would avoid a false-positive Doppler echocardiographic diagnosis of constriction in patients with chronic obstructive pulmonary disease.

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